

The NCVDLS REPORT



*Veterinary News and
Information From North
Carolina's Diagnostic
Laboratories*



North Carolina Department of
Agriculture and Consumer Services
Steve Troxler, Commissioner

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Holiday Closings...

November 11, 2015

November 26 & 27, 2015

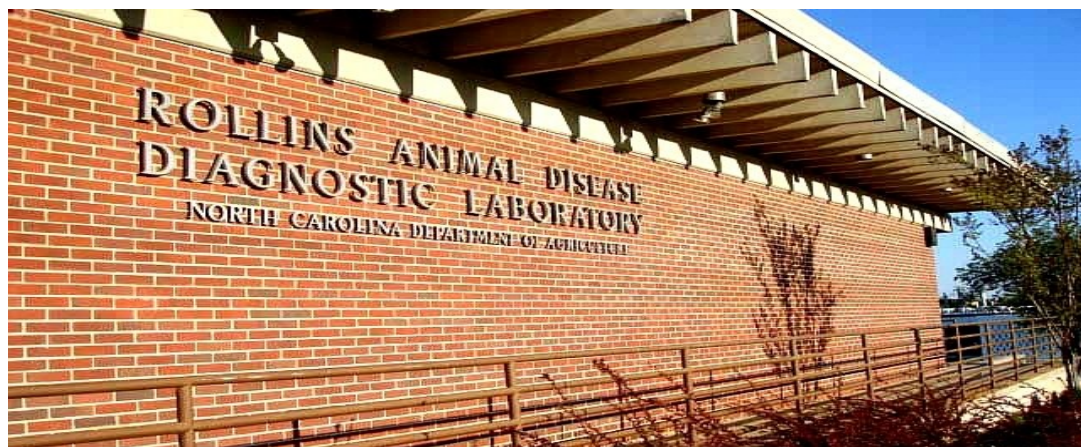
November 23, 24 & 25, 2015

Please e-mail
NCVDL@ncagr.gov with any
comments and/or suggestions
concerning The NCVDLS
Report
Editor - Dr. David Drum

Message from the Director

Two tours of the Rollins Laboratory were recently provided to legislative staff in order to garner support from the General Assembly for a \$105 million bond proposal that was added to this year's session and designated to fund a new Department of Agriculture laboratory complex in Raleigh. In addition to myself, Directors of the Food and Drug Protection and Structural Pest Divisions have met to discuss feasibility and plan for a new, state-of-the-art, co-located facility for veterinary disease diagnostic and food safety testing. This will enable the state to continue to meet the needs of NC citizens. Rapid and accurate testing capability for animal disease and food threats is a core piece of the infrastructure in supporting the nation's 8th largest animal agriculture industry, as well as, ensuring a safe, wholesome food supply and the public health of its citizens. With escalating repair and renovation costs due to aging facilities and physical space constraints, the state is lagging behind in the investment in its infrastructure that supports the core missions of protecting our consumers and our agriculture-sourced food supply. We remain hopeful as the General Assembly continues to deliberate, that the bond proposal is funded when the State's budget is finally approved.

In the wake of the nation's largest outbreak of highly pathogenic avian influenza (HPAI), the NCVDLS has been proactive to ensure laboratory preparedness and protect our State's commercial poultry (cash value of which is estimated to exceed \$5 billion) for the possibility of another outbreak associated with the wild bird and waterfowl Fall migration. The Rollins Laboratory is a member of the USDA's National Animal Health Laboratory Network (NAHLN) and has been performing the HPAI molecular-based surveillance test for quite some time. Weekly meetings with key staff were initiated to iden-



Message from the Director, *continued*

tify critical needs, develop workflow plans and testing schedules. In addition, we have secured a supply of test collection kits from the National Veterinary Services Laboratory with distribution to all facilities within our lab system. These will expedite sample collection and ultimately positively impact turnaround time for testing and permitting required by USDA to move products to market. We have been engaged with the Department's Emergency Programs Division to create a form designed specifically for sample submission and have been working with them to develop a plan to set up sample collection points throughout the state which will courier samples to the Rollins Lab. Administrative staff have been trained to ensure sample receiving and accessioning is efficient in order to expedite testing. Additional staff members have been trained to perform the polymerase chain reaction test, the official assay required by USDA for disease diagnosis. These individuals are in the process of completing their proficiency tests required by NAHLN for HPAI testing. This initiative has nearly doubled the number of available staff that can perform testing in the event of an outbreak.

Although the NCVDLs strives to provide quick turnaround and accurate, timely results to our clients, in the case of HPAI this timeliness is of a critical nature. USDA will only indemnify flock owners for live birds euthanized as part of the disease control process. There is no compensation at this time for birds that died from infection. When an outbreak of infectious disease occurs, the best method of stopping its spread is to provide rapid diagnoses necessary to prevent transmission. In this regard, we hope to continue to serve the needs of the animal health industry.

Karen W. Post DVM, MS

Feature Article**Pneumothorax in Two Dogs
Dr. Mahogany Caesar**

A 9-year-old male neutered Golden Retriever dog suddenly died while hiking. On necropsy, the diaphragm was loose, flaccid and caudally distended. The thoracic cavity was filled with abundant air and there was severe diffuse atelectasis of each lung lobe. There was moderate emphysema of the connective tissue around the trachea near the caudal cervical region and thoracic inlet. The pulmonary pleura was multifocally opaque and mildly thickened. A 2 x 1.5 cm subpleural bulla expanded the medial aspect of the right cranial lung lobe (Figure 1). Upon submersion of the pluck in water, small air bubbles exited the bulla from a centrally located rupture site that measured 3 mm in diameter (Figure 2). Atelectasis and multiple chronic pulmonary bullae were confirmed histologically.

An 11-month-old male neutered terrier mix dog was found dead in his crate. Gross examination revealed diffuse expansion of the thoracic cavity with abundant air and caudal displacement of the diaphragm. All lung lobes, except the right caudal lung lobe, were dark red to purple and severely atelectatic. The right caudal lung lobe was pink and moderately aerated with a wrinkled surface (Figure 3).

Feature Article continued

The subpleura along the medial aspect of the right caudal lung lobe was focally expanded by a 6 x 2 x 2 cm bulla. Adjacent to this focus was a 1.5 x 0.5 cm slightly depressed white focus with a centrally located 1 mm puncture surrounded by 3 x 1 cm elliptical area of hemorrhage (Figure 4). On cut section of the hemorrhagic area was an empty cavity surrounded by a thickened zone of fibrous connective tissue (fibrosis). Histopathologic examination indicated segmental pleural fibrosis with granulation tissue, segmental pulmonary interstitial fibrosis, segmental atelectasis, and segmental emphysema with bulla formation.

Both dogs died acutely from spontaneous pneumothorax secondary to a bulla rupture. Pneumothorax is the accumulation of air within the pleural space and the primary clinical sign in dogs and cats is dyspnea. It can be categorized as traumatic or spontaneous (non-traumatic). Traumatic pneumothorax is common in dogs, whereas spontaneous pneumothorax is relatively rare. Spontaneous pneumothorax most often occurs from the rupture of subpleural blebs or bullae. Other causes include bullous emphysema, bacterial pneumonia, dirofilariasis, pulmonary abscesses, paragonimus infection, and neoplasia. Pulmonary bullae and blebs occur most commonly in middle-aged, large breed, or deep-chested dogs without a previous history of respiratory disease, but they can occur in any breed and at any age. The bullae changes identified in both dogs are chronic. A prior rupture event that failed to progress to the significant degree of pneumothorax seen at the time of death in these dogs may have occurred.

From May 2011 to August 2015, 31 cases of pneumothorax have been diagnosed on necropsy at Rollins Laboratory. Species represented include 18 dogs, 7 cats, 2 cattle, 1 alpaca, 1 horse, 1 goat, and 1 pig. The following chart summarizes the causes of pneumothorax in these animals.

Number of animals or cases	Diagnoses (%)
13	Trauma (42%)
8	Undetermined/Unknown (26%)
4	Ruptured bulla (13%)
2	Parasitism (7%)
1	Bullous emphysema (3%)
1	Pulmonary neoplasia (3%)
1	Bronchopneumonia (3%)
1	Pain induced labored breathing secondary to colic (3%)

Feature Article continued

The majority (42%) of the pneumothorax cases result from a traumatic event, which includes animal attacks, fractured ribs, automobile injury, endotracheal tube placement, cardiopulmonary resuscitation efforts, and penetration by a sharp instrument. Spontaneous pneumothorax accounts for 32% of the 31 cases reported. Ruptured bulla is the leading cause of spontaneous pneumothorax and is represented in 4 dogs. Despite histopathologic examination and ancillary testing, the cause of pneumothorax is not identified in 8 cases.

Source: Lipscomb VJ, Hardie RJ, Dubielzig RR: 2003, Spontaneous pneumothorax caused by pulmonary blebs and bullae in 12 dogs. *J Am Anim Hosp Assoc* 39:435-445

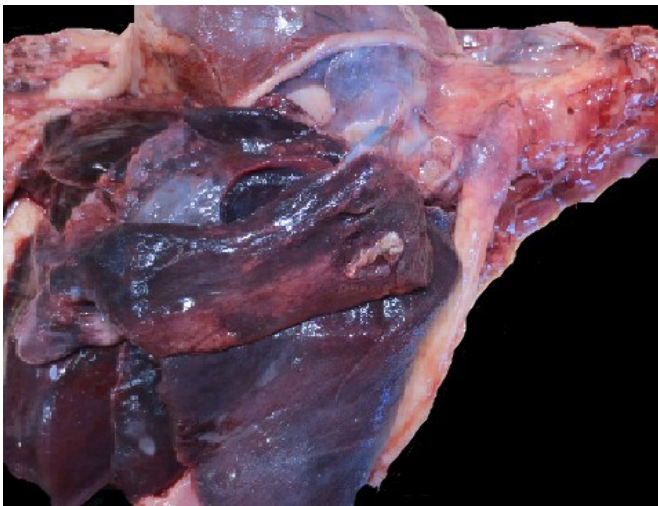


Figure 1. A 2 x 1.5 cm subpleural bulla on the right cranial lung lobe.

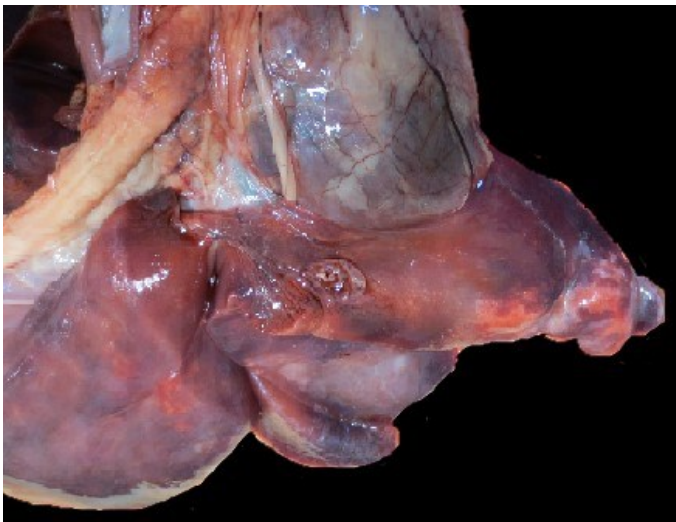


Figure 2. Infusion of the right cranial lung lobe with formalin reveals a 3 mm rupture at the central aspect of the bulla.

Feature Article continued

Figure 3. Note the aeration of the right caudal lung lobe and severe atelectasis of the remaining lobes.



Figure 4a. A 6 x 2 x 2 cm bulla expands the right caudal lung lobe. Note the 1.5 x 0.5 cm white focus with a centrally located 1 mm puncture (rupture site). A 3 x 1 cm elliptical area of hemorrhage surrounds the white focus.



Figure 4b. Cut-section of the ruptured bulla in Figure 4a.

Short Cuts

COMPANION ANIMAL

Canine

A 10 week-old, female, English Bulldog puppy collapsed and died suddenly according to the owner after eating. The owner had the puppy for about 4 weeks. The puppy had reportedly been vaccinated with its first DHPPV vaccine and dewormed at the time of purchase. The owner reported that the puppy had been healthy prior to the time of its death. On necropsy, the puppy was in ideal body condition, was mildly dehydrated, and had mild tissue autolysis present. The lungs were diffusely red to dark red in color, and were rubbery on palpation. The heart was unremarkable grossly. The liver was dark red, and mildly congested with blood. The stomach contained approximately 150-175 grams of dry dog food kibble. The intestines were slightly gas distended, and had tan colored serosal and mucosal surfaces. There was light brown, semi-liquid digesta present throughout the intestines. The colon and rectum both contained light brown, soft-formed fecal material. There were no other gross lesions in any other organ system. Histopathology showed a lymphoplasmacytic and histiocytic myocarditis which was chronic, multifocal, mild to moderate, with cardiomyocyte atrophy, degeneration, fibrosis, and rare intranuclear inclusion bodies in the heart. There was mild alveolar hemorrhage and histiocytosis in the lungs, mild eosinophilic enteritis in the intestines, a mild lymphofollicular gastritis in the stomach, and mild necrosis in the spleen. **Canine Parvovirus** was detected in the heart by immunohistochemistry. The cause of death could be attributed to the myocardial lesions, which were most consistent with a parvoviral myocarditis. The cardiac form of this disease is not common, but is usually seen in puppies 3-8 weeks of age, and can cause sudden death from cardiac arrhythmias. There typically are no intestinal lesions present in 95% of these cases.

Dr. Brad Barlow

Equine

A 4 year-old, male donkey suddenly became lethargic, and had a decreased appetite. Within 48 hours, the donkey was neurological and showing both central and peripheral neurologic signs when examined by the veterinarian on the farm. The donkey was treated with IV fluids, Banamine and Dexamethasone SP. The donkey became recumbent and was unable to stand within a few hours. Due to the severe neurological signs, and lack of response to treatment, the donkey was euthanized. On necropsy, the donkey was in good body condition, with mild dehydration and autolysis. The lungs were dark red to light gray in color, and ranged from wet and rubbery to dry and soft on palpation. The liver was diffusely dark brown, and was markedly swollen. The spleen was mild to moderately enlarged, and had a dark red to purple pulp on cross section. The stomach was markedly gas distended, and had noted ecchymosis over the serosal surface. The stomach mucosa had marked petechiation across both the glandular and non-glandular regions. The intestines had marked petechiation over the serosa/mucosal surfaces. There was dark red semi-liquid digesta present in the distal jejunum and ileum. The colon had mild to moderate petechiation over the serosal surface, and contained dark, brown feces. The urinary bladder was distended, and contained approximately 75-100mL of light red urine. The mucosa of the

COMPANION ANIMAL, CONTINUED

urinary bladder had mild to moderate petechiation. On histopathology, there was hemorrhage within the brain, severe cholestasis within the liver, tubular necrosis, with red cell casts in the kidneys, and submucosal/muscularis hemorrhage within both the stomach and small intestine. Testing for rabies, West Nile Virus, and Eastern Equine Encephalitis was all negative. The changes observed on histopathology are consistent with **Red Maple Toxicosis**. The owner had purchased the donkey just a few weeks prior to this episode and had no knowledge of potential exposure to Red Maple.

Dr. Brad Barlow

Wildlife

The body of a 16 year old female Tiger was dropped off for post mortem examination. The provided history stated the animal was euthanized that day. The tiger was not eating or drinking and was sick for 3 weeks. The tiger was not able to use its rear legs and it had an increased white blood cell count that responded slightly to antibiotics. A previous tiger from the same zoological park had similar problems.

The animal appeared to be in good body condition and there was mild post mortem change to the body overall. There was a miliary pattern of white to pale colored, slightly raised, firm circular masses throughout the lung tissue (Figure 1) . One < 1 cm wide raised, cream colored mass was present on the surface of the liver. A focal area of the pancreas was thickened. Multiple lymph nodes throughout the body (bronchiolar, epaxial and inguinal) were enlarged, misshaped and firm on palpation. The adrenal glands had an overall nodular appearance. The ovaries appeared to be enlarged and contained multiple cysts and multiple hard, solid, white colored masses of tissue. There was a 8 cm by 4.5 cm by 2 cm firm, solid, white to tan colored mass in the left rear mammary gland. No additional lesions on examination of the body

On histopathology, **Mammary gland carcinoma** was the primary neoplasia identified. Metastatic carcinoma was identified in the heart, lung, liver, lymph node, adrenal gland, and ovary. Additionally, suppurative pancreatitis and exocrine nodular hyperplasia was also identified.

Mammary carcinoma is relatively common in older large cats. There is an increased risk for cats being treated with melengestrol acetate (MGA), however, this malignancy is also seen in untreated animals. Biological behavior is aggressive with a high risk of metastatic disease to lung, liver, lymph nodes and beyond. Acute pancreatitis was also present along with nodular hyperplasia in the liver and exocrine pancreas, which are common geriatric changes.

COMPANION ANIMAL, CONTINUED

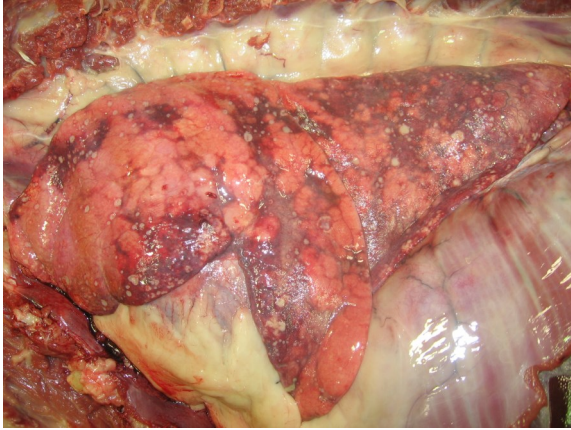


Figure 1: Extensive metastatic pulmonary tumors

Dr. David Drum

POULTRY

A 3 year old Silky cross type hen was submitted to the laboratory after having been found deceased without any indication of illness. The bird was in good body condition and the ovaries were in active production. Clotted blood filled the abdominal cavity. The spleen was severely enlarged, rounded and mottled pale tan to light red. There was a rupture in the capsule of the spleen which was the source of the hemorrhage (Figure 1).

Histopathology findings included marked deposition of amyloid like material in the spleen and pancreas.

Amyloidosis is caused by an abnormal protein material deposition in tissues and is a recognized disorder in birds and other species. Waterfowl tend to be the bird species most susceptible to amyloidosis. There is usually an underlying, predisposing disease condition such as chronic infection in birds with amyloidosis. Sudden death without observed clinical signs of disease and a lack of apparent underlying disease is not uncommon.

There is no practical treatment for amyloidosis.



Figure 1: Enlarged spleen and blood clot

Dr. Reginald Ridenhour

DEPARTMENTAL NEWS

ROLLINS LABORATORY

Employee of the Quarter

Ms. Cindy Nipper, Rollins Histology Section, Spring 2015

Rollins Lab New Hires

Amy Tyson, Medical Laboratory Technologist I, Virology Section

Danice Gregorowicz, Medical Laboratory Technologist I, Histology Section

Carla Spears, Medical Laboratory Technologist I, Virology Section

Rollins Lab Departures

Phyllis Howard, Processing Assistant IV, Administration

CE ATTENDANCE

Dr. Mahogany Caesar attended the following meetings: 2nd Annual Rabies and Other Zoonotic Diseases Course in Raleigh NC April 20-22, and the 43rd Annual Southeastern Veterinary Pathology Conference in Tifton GA May 30-31; Also she presented a case on "Intestinal and Hepatic Coccidiosis in a Rabbit".

Directory

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Director

[Dr. Karen Post](#)

Assistant Director

[Dr. Richard Mock](#)

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[Dr. James Trybus](#) (Pathology Services Coordinator)

[Dr. Tahseen Abdul-Aziz](#) (Avian)

[Dr. Allison Boone](#) (Anatomic)

[Dr. Steven Rushton](#) (Anatomic)

[Dr. Alison Tucker](#) (Anatomic)

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[Dr. Jennifer Haugland](#)

[Dr. Stacy Robinson](#)

[Dr. Mahogany Caesar](#)

Veterinary Microbiologists

[Dr. Karen Post](#)

[Dr. Richard Mock](#)

[Dr. Chad Cecil](#)

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[Sandy Murphy—Bacteriology](#)

[Mary Baker—Histopathology](#)

[Dr. Kristen Crook—Serology](#)

[Beverly Wood—Molecular Diagnostics](#)

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[Ghazala Jawad](#)

[Tina Buffington](#)

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[Dr. Reg Ridenhour](#)

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Dr. Eric Gonder	Corporate Poultry Practitioner—Goldsboro Milling
Dr. Jennifer Haugland	Veterinary Diagnostician—NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Shannon Jennings	Corporate Poultry Practitioner—Nash Johnson Farms
Dr. Randy Jones	Private Veterinary Practitioner—Livestock Veterinary Services
Dr. Richard Kirkman	Private Veterinary Practitioner—Large Animal
Dr. R. Douglas Meckes	State Veterinarian—NCDA&CS Veterinary Division
Dr. Karen Post	Director of Laboratories—NCDA&CS Veterinary Diagnostic Laboratory System
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Dr. Betsy Sigmon	Small Animal Practitioner—Creature Comforts Animal Hospital
Mr. Larry Wooten	N.C. Farm Bureau